Thermoregulatory Deficits in Zucker Rats as a Cause for Obesity

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Thermoregulatory Deficits in Zucker Rats as a Cause for Obesity. A. J. A. PRINS, M. V. D. BRINK, J. H. STRUBBE and J. POST. *Department of Animal Physiology, University of Groningen, Haren, The Netherlands.*

Permanently implanted thermocouples in liver and skin in lean Zucker rats recorded in a rise in core temperature (CT) from 38°C to a maximum 39·4°C irrespective of meal size. CT could furnish a signal to stop feeding activity. Stopping feeding between 39 and 40°C may protect liver and intestines from heat damage occurring above 40°C. Decline of peak CT while skin temperature still rose for a few minutes indicated a regulated heat flow from core to periphery. Blockade of adrenergic β-receptors with Propranolol delayed the drop of CT. Skin temperature in obese rats increased less during a meal indicating heat conservation in the core. CT of young obese rats was lower than that of lean littermates. This suggests a decreased thermogenesis which may allow larger meals to be eaten by obese rats.

Neuroendocrine Regulation of Body Fluid Homeostasis. DAVID J. RAMSAY. *Department of Physiology, University of California, San Francisco, CA 94143-0400, U.S.A.*

Numerous neuroendocrine mechanisms operate in an integrated manner to correct the hypovolemia associated with extracellular dehydration. The stimulation of the secretion of renin, aldosterone and vasopressin encourages renal salt and water conservation, and initiates drinking and/or salt appetite behavior. Cardiovascular reflexes are also involved. In situations of hypervolemia, atrial natriuretic factor (ANF) may play an important role. Atrial stretch leads to secretion of ANF, and this peptide causes inhibition of renin and aldosterone secretion under physiological conditions. Moreover, ANF inhibits baroreflex stimulated vasopressin secretion. Actions of ANF, and brain natriuretic peptide, appear to be antagonist to angiotensin II in the brain. ANF may play an important modulatory role in thirst and sodium appetite.

Post-absorptive Control of Fat Preference. DANIELLE REED, MICHAEL G. TORDOFF and MARK I. FRIEDMAN. *Monell Chemical Senses Center, Philadelphia, PA, U.S.A.*

Rats fed a High Fat diet (HFD) prefer oil more than do rats fed an isocaloric Low Fat diet (LFD). To assess whether sensory or post-absorptive effects of oil are responsible, rats with gastric cannulae were fed either the HFD and LFD. Rats drank oil for 30 min test periods while the cannula was open. The HFD group sham drank more oil than the LFD group in Trials 1–4 [$F(1,16) = 4{\cdot}8$, $p = 0{\cdot}04$] but these differences rapidly diminished. The increase in oil intake in rats fed the HFD may be due to sensory properties of oil initially but postabsorbtive factors are necessary to maintain diet-dependent differences in oil intake.

The Effect of a High Fat (HF) Diet on Food Intake and Eating Behavior in the Rat. A. RICHARDSON, R. D. E. RUMSEY and N. W. READ. *Sub-department of GI Physiology and Nutrition, The University, Sheffield, U.K.*

Feeding behaviour was continuously monitored in rats fed a normal diet, with a normal fat content (4·8%). The effect of feeding behaviour of changing the diet from normal fat content to HF content (30%) for 6 days was determined. After 6 days the effect of returning to a normal diet was assessed for a further 10 days. The effect of the HF diet was marked but transient hyperphagia, due to an increase in average meal size and rate of eating without altering average meal frequency. After 2 days on the HF diet, the normal calorie control of food intake was restored. Returning to the normal diet produced a significant transient hypophagia, due to decreased meal size. Food intake and eating behaviour returned to normal after 2 days.